CDC Home | Search | Health Topics A-Z

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Chagas Disease After Organ Transplantation --- Los Angeles, California, 2006

Chagas disease is an infection caused by the parasite *Trypanosoma cruzi*. Reduviids (i.e., "kissing bugs") transmit the parasite through infected feces. *T. cruzi* also can be transmitted congenitally and through blood transfusion or organ transplantation. The infection is lifelong if left untreated; the majority of infected persons are asymptomatic, and their disease remains undiagnosed. Although routine serologic testing of organ and blood donors is performed in areas of Latin America where Chagas disease is endemic, no *T. cruzi* screening test is licensed in the United States. However, seroprevalence studies using research tests have documented the presence of *T. cruzi* antibodies in U.S. blood (*1*) and organ donor populations (*2*). This report describes two cases of acute Chagas disease in heart transplant recipients reported by two Los Angeles County hospitals in February 2006. In the United States, one previous report documented *T. cruzi* transmission through solid organ transplantation, in which three organ recipients were infected (*3*).

Case Reports

Case 1. In December 2005, a man aged 64 years with idiopathic cardiomyopathy received a heart transplant. In January 2006, he was treated with enhanced immunosuppression for suspected organ rejection. In February 2006, he was readmitted to the hospital with anorexia, fever, and diarrhea of 2 weeks' duration. A peripheral blood smear revealed *T. cruzi* trypomastigotes, blood cultures were positive for *T. cruzi*, and endomyocardial biopsy specimens contained amastigotes. The patient was interviewed about natural exposures, and organ procurement and transplantation records were reviewed. He had no identifiable risk factors for *T. cruzi* infection (e.g., travel to a country endemic for Chagas disease). He was seronegative for *T. cruzi* antibodies but positive for *T. cruzi* DNA by polymerase chain reaction (PCR), indicating recent infection. After initiation of nifurtimox therapy, his parasitemia rapidly cleared. However, in April 2006, the patient died from complications attributed to acute rejection of the transplanted organ.

To identify the source of infection, a traceback was conducted on all blood products transfused to the heart donor and recipient. All available blood donors tested negative for *T. cruzi* antibodies by immunofluorescence assay (IFA) and radioimmunoprecipitation assay (RIPA). However, blood from the organ donor tested seropositive for *T. cruzi* antibodies by RIPA and tested borderline-positive by IFA. The organ donor had been born in the United States but had traveled to a *T. cruzi*--endemic area of Mexico.

Three additional patients received a liver and both kidneys from the same donor. These patients are *T. cruzi*--seronegative by IFA and have no evidence of parasitemia by PCR. They continue to be monitored.

Case 2. In January 2006, a man aged 73 years with ischemic cardiomyopathy received a heart transplant. The patient was readmitted to the hospital in February 2006 with fever, fatigue, and an abdominal rash. A thin blood smear revealed *T. cruzi* trypomastigotes, and blood cultures were positive for *T. cruzi*. Organ procurement and transplantation records were reviewed. The patient had no identifiable risk factors for *T. cruzi* infection. He was seronegative but PCR-positive

for *T. cruzi*, indicating recent infection.

The patient's rash and parasitemia resolved after 10 days of nifurtimox treatment. Serial endomyocardial biopsies did not reveal trypanosomes, and he remained seronegative by IFA for *T. cruzi*. The patient died in June 2006. The primary cause of death was cardiac failure; no autopsy was performed.

The source of infection was investigated with the same methods used for case 1. All available blood donors tested seronegative for *T. cruzi*. The organ donor, who had been born in El Salvador and was residing in Los Angeles at the time of his death, tested positive for *T. cruzi* antibodies by RIPA but had a negative IFA. Three other patients received solid organs from the same donor. These patients are *T. cruzi*-seronegative by IFA and have no evidence of parasitemia by PCR. They continue to be monitored. No record of previous blood donations by either organ donor was found.

Reported by: *L Mascola, MD, Acute Communicable Disease Control Program, Los Angeles Dept of Health Svcs; B Kubak, MD, Univ of California; S Radhakrishna, MD, Univ of Southern California; T Mone, One Legacy, Los Angeles; R Hunter, California Dept of Health Svcs. DA Leiby, PhD, American Red Cross, Rockville, Maryland. M Kuehnert, MD, Div of Healthcare Quality Promotion, National Center for Infectious Diseases; A Moore, MD, F Steurer, MS, G Lawrence, MPH, Div of Parasitic Diseases, National Center for Preparedness, Detection, and Control of Infectious Diseases (proposed); H Kun, ScD, EIS Officer, CDC.*

Editorial Note:

The two cases described in this report are the fourth and fifth cases of reported *T. cruzi* transmission through solid organ transplantation in the United States. The prevalence of infection with *T. cruzi* in the United States varies by region and might now be higher than previously thought, especially in geographic areas such as Los Angeles County, where a substantial proportion of blood and organ donors have emigrated from Chagas-endemic countries. Because organ donors frequently receive blood transfusions, infection can be transmitted to recipients either by transfusion or transplant. Currently, no policies recommend laboratory screening for *T. cruzi*. Diagnostic tests available for research studies have variable sensitivities and specificities, and no licensed screening test exists.

Physicians and laboratorians should maintain a high index of suspicion for *T. cruzi* infection in transplant and transfusion recipients who exhibit complications of an unknown etiology when more common sources have been excluded. Acute Chagas disease in severely immunocompromised patients is of special concern because the clinical course is often severe and rapidly progressive. If Chagas is suspected, manual microscopic examination of peripheral blood smears should be performed. Patients with acute Chagas disease should be treated as early as possible in the course of the infection. Available treatments include nifurtimox (available from CDC Drug Service, telephone 404-639-3670) or benznidazole (only distributed outside of the United States).

References

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3. CDC. Chagas disease after organ transplantation---United States, 2001. MMWR 2002;51:210--2.

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